

CASE REPORTS

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Endocarditis Lenta Due to *Staphylococcus Aureus*

Report of a Case

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THIS report of a single case of *staphylococcus aureus* endocarditis with penicillin treatment and clinical arrest is made in view of certain difficulties involved in the diagnosis and because of the mild afebrile course.

The only similar case found in the literature was one reported by MacNeal,² in which a patient with *staphylococcus aureus* endocarditis had low-grade fever, never over 100° F. except on one occasion after reaction to penicillin. In that case the patient had intermittent courses of treatment with penicillin over a period of several months. The amount of penicillin given, which was governed by the amount then (1944) available, ranged from 500 units to 10,000 units every two hours. The patient was finally "cured" when the article was published a year later.

In their section on bacterial endocarditis in "Oxford Medicine," Libman and Friedberg¹ stated that "*staphylococcus aureus* has been reported as the cause of subacute bacterial endocarditis, but we have no confirmatory experience." They go on to discuss "mild cases of bacterial endocarditis," but they do not mention *staphylococcus aureus* as an etiological agent.

The patient in the present case had mild bacterial endocarditis. With regard to this case, however, "lenta" is considered a more exact word than "mild," because "lenta" implies a slow course. Before the advent of antibiotics, almost all bacterial endocarditis was fatal and it was difficult to convince members of a family that the patient had "mild endocarditis" when in the next breath it was explained that the outcome was likely to be fatal. On the other hand, it seems reasonable that "slow" endocarditis can terminate fatally.

CASE REPORT

A 72-year-old man was admitted to Franklin Hospital, San Francisco, on March 3, 1946, complaining of generalized arthralgia and weakness of one year's duration. He had been well until about one year before admission, at which time he noted the onset of "severe low-back pain" which had been attributed by a physician to a recent attack of "flu." The pain gradually increased and spread throughout the spine. About ten months prior to admission the patient began to lose weight, and the pain spread to the limbs. It became so severe that the patient was unable to dress himself.

About seven months prior to admission the hemoglobin value was found to be 60 per cent. Iron, liver extract, and

blood transfusions did not relieve the anemia. On admission to Franklin Hospital, the complaints were severe generalized pain in the back, made worse by motion, and severe pain in both arms, both shoulders, and both wrists. The pain was so severe as to interfere with sleep, and the patient was hardly able to get about his room. Any activity made the pain worse. There had been a loss of about 40 pounds in weight in ten months. The patient said he had not had chills or fever.

Past History: In childhood the patient had had Osgood-Schlatter disease. About 30 years before the present illness he had had "blood poisoning" of the left hand, followed by a "continuous siege of boils for several years." About 25 years ago "severely infected" tonsils had been removed, and this seemed to "cure the boils." Twenty years ago cholelithiasis had developed and cholecystectomy was done. About ten years ago the patient had noted angina pectoris on exertion and was told his blood pressure was "250." He had never noted edema or orthopnea, and he said that so far as he knew he had not had rheumatic fever. His first knowledge of cardiac murmur was about one year ago.

Physical Examination: The patient was well-developed but underweight. The general appearance was in accord with the stated age of 72. The patient was in extreme distress and did not seem comfortable in any position. There was a generalized *cafe au lait* tint to the skin, more pronounced over the chest. Several small pigmented moles were observed but no petechiae or other rash was noted. There was no apparent focus of infection. The chest was emphysematous with a few coarse, moist rales at both bases. The heart sounds were distant. A high-pitched, rough systolic murmur was heard over the whole precordium. This murmur was loudest at the apex and was transmitted to the axilla but not to the back. A soft, blowing systolic murmur was heard in the aortic area. The abdomen was slightly distended and tympanitic. The liver edge was 7 cm. below the right costal margin and was firm, smooth, and non-tender. The tip of the spleen was 4 cm. below the left costal margin; its edge was rounded, hard, and non-tender. The fingers were cyanotic and clubbed, but no splinter hemorrhages were seen. The reflexes were equal and active throughout. Otherwise, the physical examination was essentially normal.

Laboratory Work: There was a slight trace of albumin but no erythrocytes in the urine. The hemoglobin content in the blood was the equivalent of 10 gm. per 100 cc. Erythrocytes numbered 4,030,000 and leukocytes 6,200, with 60 per cent polymorphonuclear cells, 35 per cent lymphocytes, 4 per cent monocytes, and 1 per cent basophils. Results of Wassermann and Kahn tests were negative for syphilis. Total blood protein content was 8.21 gm. per 100 cc.

An electrocardiogram showed a pulse rate of 82, with normal rhythm and occasional premature beats; the P-R interval was 0.16 second; and the QRS interval was 0.08 second; T3 was diphasic.

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Blood cultures revealed the following: March 3, 1946: Positive for staphylococcus aureus.

March 7, 1946: Positive for staphylococcus aureus.

March 8, 1946: Positive for staphylococcus aureus. Plate count showed 25 colonies per cc. The organisms were sensitive to penicillin.

March 10, 1946: Positive for staphylococcus aureus.

March 15, 20, 25, and 30, 1946: Negative.

April 10, 20, and 30, 1946: Negative.

Treatment consisted of penicillin, 20,000 units every two hours, from March 10 to March 19, 1946. Then, from March 19 to April 17, 1946, 25,000 units was given every three hours. The total was 7,960,000 units in about five weeks.

Throughout a six-week stay in the hospital the patient was afebrile. The pulse rate ranged from 90 to 100 per minute, and respirations from 20 to 24 per minute.

By June 1946, three months after treatment was started, the patient had regained about two-thirds of the weight he had lost and was almost completely relieved of "arthritic pains." He had returned to his office where he worked several hours each day.

Now, three years later, the patient is working in his office ten to twelve hours daily. There has been no change in the cardiac murmur and no evidence of congestive failure. The "arthritic pains" are gone and blood cultures have shown no growths.

DISCUSSION

In this case an interesting problem of diagnosis was presented. The patient was seen in consultation primarily because of anemia which was believed to be the cause of

weakness. The enlarged spleen gave an important clue which, added to the findings of clubbed fingers, *cafe au lait* pigmentation, and cardiac murmur, caused endocarditis to be considered. It was surprising to find staphylococcus aureus as the offending organism, but repeated studies confirmed this finding. It is interesting to speculate as to the date of onset of septicemia, for it may well have started with the onset of the "arthritis" a year before. The fact that the "arthritic pains" ceased following treatment suggests that they were related to the septicemia. Afebrile staphylococcus-aureus septicemia of any duration is extremely rare. The relatively small doses of penicillin given appear to have been adequate. However, in light of recent experience, larger doses probably would be given now in similar circumstances.

SUMMARY

A case of afebrile staphylococcus-aureus endocarditis lenta with penicillin treatment and clinical arrest of three years is reported. Only one similar case has been reported in the literature. The importance of blood cultures, even in the absence of fever, is again demonstrated.

REFERENCES

1. Libman, E., and Friedberg, C. F.: Subacute bacterial endocarditis, Oxford Medicine (Oxford University Press), Vol. II, page 346.
2. MacNeal, W. J., Poindexter, C. A., and Martey, F. N.: Apparent arrest of staphylococcal endocarditis, Am. Heart J., 29:403-408, March 1945.

Allergic Reaction to Decholin Used in Circulation Test

Report of a Case

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THE value of the circulation time as an aid in the determination of the presence of congestive heart failure has been well established.^{2,4} Decholin® (sodium dehydrocholate) was introduced into medicine for this purpose in 1931 by Winternitz.⁵ Other compounds such as calcium gluconate have been used for the same purpose but the latter has a synergistic action with digitalis and is potentially toxic when used in digitalized patients. Decholin, on the other hand, is relatively non-toxic and the only contraindications to its use as stated in a brochure prepared by the manufacturer* are: (1) obstructive jaundice, (2) bronchial asthma.

Untoward reactions to Decholin are rare. A search of the American literature reveals only a report by Norman³ in 1947 of three cases in which allergic reactions occurred subsequent to the injection of Decholin. In none of these cases was there a clear-cut history of previous allergic disease. In one case there was no reaction to the first injection of Decholin but a second injection one week later was followed by shock. In another case, injection was followed in one minute by a violent asthmatic attack. (In this instance, a review of the clinical findings suggests that the dyspnea was probably due to unrecognized asthmatic bronchitis.) In the third case, widespread diffuse urticaria developed five minutes after the injection of Decholin.

The following case report is submitted as the fourth case in which frank allergic manifestations occurred following the parenteral use of Decholin.

CASE REPORT

The patient, a 52-year-old white woman, was first observed April 12, 1949, for evaluation of hemoptysis. X-ray films of the chest made at this time revealed healed apical pulmonary tuberculosis ("fibrocalcific scarring but no evidence of active parenchymal disease"), bilateral basal bronchiectasis, and generalized pulmonary emphysema. A study of the sputa on several occasions did not reveal Koch's bacilli. There had been previous attacks of hemoptysis in 1918, 1938, and 1942. There was no record of previous allergic manifestation. In 1942 the patient had had a "heart attack," characterized by substernal oppression in the xiphoid area and pain along the left anterior costal margin with pronounced tachycardia. Electrocardiograms made during this attack were not available but an electrocardiogram made during the present study revealed a left bundle branch block. Slight left ventricular enlargement was noted in an x-ray film of the chest. The amount of bloody sputum decreased sharply during a six-day stay in the hospital, and the patient was discharged April 18, 1949.

In order to decrease the amount of purulent sputum, inhalations of penicillin dust were taken at home, but after several days a local sensitization developed in the form of a sore throat.

On May 16, 1949, the patient complained of dyspnea on recumbency, together with headache. Both symptoms were relieved only by sitting up repeatedly during the night. Physical examination revealed the lungs to be clear. The heart findings included a soft systolic murmur which was Grade 2 at the apex and was transmitted to the axilla. The systolic murmur was Grade 1 at the base. The aortic second sound was not accentuated. The rhythm was regular. The blood pressure was 155 mm. of mercury systolic and 95 mm. diastolic. The liver was not enlarged. No edema of the lower extremities was discernible nor was there visible distention of the peripheral veins of the extremities.

*Ames, Inc., Elkhart, Ind.